U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 20 November 2006

In the Matter of

E.M.C.[1], widow of
F.C., Jr.
Claimant

Case No.: 2005 BLA 77

v.

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party in Interest

Appearances: Mr. J.D.C., Lay Representative and son of Mrs. E.M.C.
For the Claimant

Ms. Suzanne F. Dunne, Attorney
For the Director

Before: Richard T. Stansell-Gamm
Administrative Law Judge

DECISION AND ORDER --
APPROVAL OF MODIFICATION REQUEST &
AWARD OF SURVIVOR BENEFITS

This matter involves a claim filed by Mrs. E.M.C. for survivor benefits under the Black Lung Benefits Act, Title 30, United States Code, Sections 901 to 945 ("the Act"). Benefits are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis, or to survivors of persons who died due to pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as "black lung" disease.

[1]Despite 20 C.F.R. § 725.477(b) ("A decision and order shall contain . . . the names of the parties . . . ."), and over my specific objection, Chief Administrative Law Judge John Vittone has directed that I substitute initials for the names of the Claimant and all family members. Any comments or concerns regarding this mandated practice should be directed to Chief Administrative Law Judge John Vittone, 800 K Street, Suite 400N, Washington, D.C. 20001.

Evidentiary Comment

At the August 2005 hearing, I admitted into evidence the documents previously marked as DX[2] 1 to DX 55. Upon adjudication of this claim, I discovered an additional folder marked "Non-Evidence" by the Director. The folder consists mainly contains additional correspondence by the Claimant's lay representative, prior hearing transcripts, administrative letters and transmittal actions. However, some of the documents help explain the procedure history of this case. As a result, I now admit the folder as DX 56.

Procedural Background

Mr. F.C.'s Black Lung Disability Claim
(DX 20)

On July 8, 1988, Administrative Law Judge Stuart A. Levin approved Mr. C.'s claim for black lung disability benefits. During the proceeding, the Director's representative agreed that Mr. C. had pneumoconiosis and was totally disabled. The remaining issue was whether Mr. C.'s totally disabling respiratory impairment was due to pneumoconiosis. In the medical opinion Judge Levin found more probative, the physician concluded that Mr. C. was exposed to three pulmonary risks, listed in order of importance: cigarette smoking, silica dioxide exposure in a foundry, and coal mine dust exposure during four and a half years of coal mine employment. Since the physician believed the three factors had a cumulative effect, he attributed Mr. C.'s pulmonary disability in part to his coal mine employment.

Mrs. E.C.'s Survivor Claim

Initial Adjudication

On October 21, 1996, Mrs. C. filed her claim for survivor benefits under the Act (DX 1). On February 28, 1997, the Director denied Mrs. C.'s claim for failure to prove that her husband's death was due to coal workers' pneumoconiosis (DX 7). On March 21, 1997, Mrs. C. appealed the denial of her claim and submitted additional medical documentation (DX 9). Upon additional consideration, the Director again denied Mrs. C.'s claim on April 3, 1997 (DX 10). Again, on April 14, 1997, Mrs. C. appealed (DX 11). On June 30, 1997, July 2, 1997, and September 2, 1997, Mrs. C. submitted a statement from a family doctor indicating pneumoconiosis was a factor in Mr. C.'s death and three hundred pages of medical studies and articles to support her contention that the presence of crystalline silica in respirable coal mine dust contributed to Mr. C.'s development of lung cancer and hastened his death (DX 13, DX 14, and DX 15). On September 24, 1997, following an informal conference, the Director denied Mrs. C.'s claim, concluding coal workers' pneumoconiosis did not cause or contribute to her husband's death (DX 17). Mrs. C. appealed and the case was forwarded to the Office of Administrative Law Judges ("OALJ") on October 10, 1997 (DX 18 and DX 21).

[2]The following notations appear in this decision to identify exhibits: DX – Director exhibit; CX – Claimant exhibit; ALJ – Administrative Law Judge exhibit; and TR – Transcript.

First Administrative Law Judge Decision
(DX 24)

After conducting a hearing in March 1998, Administrative Law Judge Gerald M. Tierney denied Mrs. C.'s survivor claim on September 23, 1998 principally due to the absence of any opinion by a physician indicating the Mr. C.'s lung cancer was due in part to his exposure to coal mine dust. Judge Tierney also noted the absence of any medical evidence indicating Mr. C.'s lung damage was caused by exposure to silica. Finally, after indicating the treating physician's opinion was not sufficiently reasoned and finding another medical expert's analysis probative, Judge Tierney concluded coal workers' pneumoconiosis did not cause or hasten Mr. C.'s death due to lung cancer.

First Modification Request
(DX 25 to DX 32, and DX 56)

On October 13, 1998, Mrs. C. appealed Judge Tierney's adverse decision to the Benefits Review Board ("BRB" and "Board"). To support her appeal, Mrs. C. subsequently sent an additional medical report. In response the BRB advised Mrs. C. that they could not consider evidence not already in the record. When the Claimant nevertheless asked the BRB to consider the new evidence, the Board deemed the correspondence to be a request for modification and return the record to the Director for modification proceedings on December 29, 1998. On January 18, 1999, the Director informed Mrs. C.'s lay representative that the additional information would be considered as a request for modification. On April 2, 1999, upon consideration of the additional information provided by Mrs. C., the Director determined modification of Judge Tierney's denial of benefits was not warranted. On April 12, 1999, Mrs. C. appealed the decision and requested a hearing. The case was forwarded to OALJ on May 5, 1999.

Second Administrative Law Judge Decision
(DX 35 and DX 56)

On March 23, 2000, Administrative Law Judge Robert L. Hillyard conducted a second hearing. On June 30, 2000, Judge Hillyard denied Mrs. C.'s modification request. For various reasons, Judge Hillyard found insufficient probative medical opinion to establish that a mistake of fact had been made by Judge Tierney. Based on the preponderance of probative medical opinion, Judge Hillyard concluded Mr. C. did not die due to pneumoconiosis. On July 25, 2000, Mrs. C. appealed the denial of her claim.

Benefits Review Board Decision
(DX 36)

On July 30, 2001, the BRB affirmed Judge Hillyard's denial of Mrs. C.'s modification request and claim for survivor benefits.

Second Modification Request
(DX 37 to DX 46)

On September 13, 2001, Mrs. C. filed a second modification request. On October 2, 2002, the Director closed the modification proceeding due to inactivity. On October 14, 2002, Mrs. C. objected to the Director's action and requested continued adjudication of her modification request. On January 29, 2003, the case was returned to OALJ.

Third Administrative Law Judge Decision
(DX 51 and DX 56)

On June 20, 2003, Administrative Law Judge Edward Terhune Miller conducted a third hearing in this case. On October 6, 2004, Judge Miller denied the modification request and claim for survivor benefits. Judge Miller concluded Mrs. C. had presented probative medical opinion to support a finding that silica in coal mine dust may cause lung cancer. However, due to the absence of objective medical evidence, Judge Miller concluded the record remained insufficient to establish that Mr. C.'s short exposure to coal mine dust caused or contributed to his development of lung cancer. On January 24, 2005, upon reconsideration of his probative value determinations, Judge Miller concluded his original findings remained valid.

Third Modification Request and Present Proceeding
(DX 53 to DX 55)

On February 2, 2005, Mrs. C. filed her third modification request. On July 1, 2005, the request was forwarded to OALJ for a determination. Pursuant to a Notice of Hearing, dated August 25, 2005 (ALJ I), I conducted a hearing in Carbondale, Illinois on October 26, 2005. My decision in this case is based on DX 1 to DX 56 and CX 1.

ISSUES

1. Whether in filing a modification request on February 2, 2005, Mrs. C. demonstrated that a mistake in determination of fact occurred in Judge Miller's denial of her second modification request on January 24, 2005.

2. If Mrs. C. establishes a change in condition or a mistake of fact, whether Mr. C.'s death was due to coal workers' pneumoconiosis.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Stipulations of Fact

At the October 26, 2005 hearing, the parties stipulated: a) Mr. C. had at least four and a half years of coal mine employment; b) Mr. C. had coal workers' pneumoconiosis; and, c) Mrs. C. is an eligible survivor under the Act. (TR, p. 13-14).

Preliminary Findings

Born on December 12, 1928, Mr. C. married Mrs. C. on August 31, 1947. Between 1945 and 1949, Mr. C. worked as a coal miner. Additionally, between 1951 and 1965, Mr. C. worked in a magnesium factory. He also was employed an additional twelve years in a lumber yard. Mr. C. smoked a pack of cigarettes for 40 years before quitting in 1995.[3] On October 4, 1996, Mr. C. passed away. (DX 1 to DX 4, DX 8, and DX 20).

Issue # 1 – Modification

Any party to a proceeding may request modification at any time before one year from the date of the last payment of benefits or at any time before one year after the denial of a claim. 20 C.F.R. § 725.310(a). Upon the showing of a "change in conditions" or a "mistake in a determination of fact" the terms of an award or the decision to deny benefits may be reconsidered. 20 C.F.R. § 725.310. An order issued at the conclusion of a modification proceeding may terminate, continue, reinstate, increase or decrease benefit payments or award benefits.

Since the present modification relates to Mrs. C.'s survivor claim, evaluation of the record for a change in conditions is not warranted.[4] Instead, the focus in modification proceedings in a survivor claim concerns a mistake of fact analysis. In *O'Keefe v. Aerojet-General Shipyards, Inc.*, 404 U.S. 254, 257 (1971), the United States Supreme Court indicated that an administrative law judge should review all evidence of record to determine if the original decision contained a mistake in a determination of fact. In considering a motion for modification, the administrative law judge is vested "with broad discretion to correct mistakes of fact, whether demonstrated by wholly new evidence, cumulative evidence, or merely further reflection on the evidence initially submitted." *See also Jessee v. Director, OWCP*, 5 F.3d 723 (4th Cir. 1993); *Director, OWCP v. Drummond Coal Co. [Cornelius]*, 831 F.2d 240 (11th Cir. 1987).

My determination of whether a mistake of fact occurred during the prior adjudication of Mrs. C.'s survivor claim involves the four entitlement elements that a claimant must prove by a preponderance of the evidence to receive survivor benefits under the Act and 20 C.F.R. § 718.205(a). The claimant bears the burden of establishing these elements by a preponderance of the evidence. If the claimant fails to prove any one of the requisite elements, the survivor claim for benefits must be denied. *Gee v. W. G. Moore and Sons*, 9 B.L.R. 1-4 (1986) and *Roberts v. Bethlehem Mines Corp*., 8 B.L.R. 1-211 (1985).

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[3]Mr. C.'s reported smoking histories varied from less than 25 years to more than 75 packs years (a pack year equals the consumption of a pack of cigarettes a day for one year). Further, several friends and co-workers opined that he smoked less than a pack a year and for less than 40 years (DX 39). However, based on the predominant summaries by most of the physicians who reviewed his case and Mr. C.'s treatment notes which reflect his stated smoking history, I find Mr. C. smoked a pack of cigarettes a day for 40 years.

[4]Since Mr. C. has passed away, there can be no change in conditions concerning his pulmonary condition since the prior denials of Mrs. C.'s survivor claim.

First, the claimant must establish eligibility as a survivor. A surviving spouse may be considered eligible for benefits under the Act if she was married to, and living with, the coal miner at the time of his death, and has not remarried.[5]

Second, the claimant must prove the coal miner had pneumoconiosis.[6] "Pneumoconiosis" is defined as a chronic dust disease arising out of coal mine employment. The regulatory definitions include both clinical pneumoconiosis (the diseases recognized by the medical community as pneumoconiosis) and legal pneumoconiosis (defined by regulation as any chronic lung disease arising out of coal mine employment).[7] The regulation further indicates that a lung disease arising out of coal mine employment includes "any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment."[8] As courts have noted, under the Act, the legal definition of pneumoconiosis is much broader than medical pneumoconiosis. *Kline v. Director, OWCP*, 877 F.2d 1175 (3d Cir. 1989).

Third, once a determination has been made that a miner had pneumoconiosis, it must be determined whether the coal miner's pneumoconiosis arose, at least in part, out of coal mine employment.[9]

Fourth, the surviving spouse has to demonstrate the coal miner's death was due to pneumoconiosis.[10]

Mrs. C.'s third modification request relates to the fourth element of entitlement and requires an evaluation of the record to determine whether a mistake of fact occurred in Judge Miller's decision that Mr. C.'s death was not caused by coal workers' pneumoconiosis. Mrs. C. asserts a mistake of fact occurred in two ways. First, due to Mr. C.'s exposure to coal mine dust containing a known carcinogen, crystalline silica, his terminal lung cancer represented a complication of pneumoconiosis which caused his death. Second, Mr. C.'s coal workers' pneumoconiosis was a contributing cause of Mr. C.'s death.

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[5]20 C.F.R. § 718.4 indicates that the definitions in 20 C.F.R. § 725.101 are applicable. 20 C.F.R. § 725.101, in turn, refers to the term "survivor" as used in Subpart B of Part 725. 20 C.F.R. § 725.214 then sets out the spousal relationship requirements and 20 C.F.R. § 725.215 describes the dependency rules. According to § 725.214(a) the spousal relationship exists if the relationship is a valid marriage under state law. Under § 725.215(a), a spouse is deemed dependent if she was residing with the miner at the time of his death.

[6]20 C.F.R. § 718.205(a)(1); *see Trumbo v. Reading Anthracite Co*., 17 B.L.R. 1-85 (1993).

[7]20 C.F.R. § 718.201(a)(1) and (2).

[8]20 C.F.R. § 718.201(b).

[9]20 C.F.R. §§ 718.203(a) and 205(a)(2).

[10]20 C.F.R. § 718.205(a)(3).

Death Due to Pneumoconiosis

For a survivor claim filed on or after January 1, 1982, the Department of Labor regulations provide four means by which to establish that a coal miner's death was due to coal workers' pneumoconiosis:[11]

1. The miner had complicated pneumoconiosis;

2. Death was caused by pneumoconiosis;

3. Death was caused by complications of pneumoconiosis; or,

4. Pneumoconiosis was a substantially contributing cause or factor leading to the miner's death.

However, a survivor may not receive benefits if the coal miner's death was caused by traumatic injury, or the principal cause of death was a medical condition not related to pneumoconiosis, unless evidence establishes that pneumoconiosis was a substantially contributing cause of death.

Complicated Pneumoconiosis

In the Black Lung Benefits Act, 30 U.S.C. 921 (c) (3) (A) and (C), as implemented by 20 C.F.R. § 718.304(a), Congress determined that if a miner is suffering from a chronic dust disease of the lung "which when diagnosed by chest roentgenogram, yields one or more large opacities (greater than one centimeter in diameter)[12] and would be classified in category A, B, or C . . . there shall be an irrebuttable presumption that . . . his death was due to pneumoconiosis." This type of large opacity is called "complicated pneumoconiosis." The statute and 20 C.F.R. §§ 718.304(b) and (c) also permit complicated pneumoconiosis to be established by either the presence of massive fibrosis in biopsy and autopsy evidence or other means which would be expected to produce equivalent results in chest x-rays or biopsy/autopsy evidence.

All evidence relevant to whether the miner has complicated pneumoconiosis must be weighed. *Melnick v. Consolidation Coal Co.,* 16 B.L.R. 1-31 (1991); *Maypray v. Island Creek Coal Co.*, 7 B.L.R. 1-683 (1985). Therefore, even after the presence of large opacities have been established through one of the methods set out in § 718.304, all other medical evidence must be considered and evaluated to determine if relevant evidence conflicts with or confirms a finding

[11]20 C.F.R. §§ 718.205(c)(1), (2), and (3), and 304.

[12]On the standard ILO chest x-ray classification worksheet, Form CM 933, large opacities are characterized by three sizes of opacities, identified by letters. The interpretation finding of Category A indicates the presence of a large opacity having a diameter greater than 10 mm (one centimeter) but not more than 50 mm; or several large opacities, each greater than 10 mm but the diameter of the aggregate does not exceed 50 mm. Category B mean an opacity, or opacities, "larger or more numerous than Category A" whose combined area does not exceed the equivalent of the right upper zone of the lung. Category C represents one or more large opacities whose combined area exceeds the equivalent of the right upper zone.

of large opacities and an association with pneumoconiosis. For example, the Benefits Review Board affirmed a finding of complicated pneumoconiosis under 20 C.F.R. § 718.304 when the administrative law judge considered chest x-rays in conjunction with CT scan findings to determine there was sufficient evidence to find complicated pneumoconiosis. *Keene v. G&A Coal Co.*, BRB No. 96-1689 BLA (Sept. 27, 1996).

In light of these statutory, regulatory and judicial principles, the adjudication of whether a claimant is able to invoke the irrebuttable presumption under 20 C.F.R. § 718.304 involves a two-step process. First, I must determine whether: a) the preponderance of the chest x-rays establishes the presence of large opacities characterized by size as Category A, B, or C under recognized standards; or b) biopsy evidence or other diagnostic results exist which are equivalent to chest x-ray evidence of large opacities characterized as Category A, B, or C. At this stage of the process, the essential inquiry is whether such large opacities, or their equivalent, exist.

Second, if the preponderance of the evidence does demonstrate the existence of large opacities, I must then consider all other relevant evidence to determine whether that evidence contradicts or supports a finding that the large opacities are both present and indicative of complicated pneumoconiosis.

Existence of Large Opacities

To demonstrate the presence of a large pulmonary opacity, Mrs. C. may rely on chest x-ray imaging, biopsy, or other medical tests, such as CT scans, showing the equivalent of a radiographic image.

Chest X-Rays

Date of x-ray	**Exhibit**	**Physician**	**Interpretation**
Oct. 15, 1973	DX 6	Dr. Pozsgay	Mild chronic fibrosis and emphysema; no active lung disease.
Jan. 24, 1976	DX 6	Dr. Shippey	Essentially normal.
Nov. 21, 1978	DX 6	Dr. Shippey	Very mild emphysema with minimal, non-specific fibrosis.
Apr. 12, 1980	DX 6	Dr. Shippey	Non-specific fibrosis.
Sep. 4, 1985	DX 49	Dr. Hummel	Positive for pneumoconiosis, profusion category 1/1,[13] type s opacities.[14] No large opacity noted.

[13]The profusion (quantity) of the opacities (opaque spots) throughout the lungs is measured by four categories: 0 = small opacities are absent or so few they do not reach a category 1; 1 = small opacities definitely present but few in number; 2 = small opacities numerous but normal lung markings are still visible; and, 3 = small opacities very numerous and normal lung markings are usually partly or totally obscured. An interpretation of category 1, 2, or 3 means there are opacities in the lung which may be used as evidence of pneumoconiosis. If the interpretation is 0, then the assessment is not evidence of pneumoconiosis. A physician will usually list the interpretation with two digits. The first digit is the final assessment; the second digit represents the category that the doctor also seriously considered. For example, a reading of 1/2 means the doctor's final determination is category 1 opacities but he considered placing the interpretation in category 2. Additionally, according to 20 C.F.R. § 718.102(b), a profusion reading of 0/1 does not constitute evidence of pneumoconiosis.

(same)	DX 49	Dr. Sargent, BCR, B[15]	Positive for pneumoconiosis, profusion category 1/0, type t/q opacities. No large opacities noted.
(same)	DX 49	Dr. Britton, A	Positive for pneumoconiosis or silicosis, profusion category 1/0, type q/p opacities. Questionable mass upper right chest; however, pneumoconiosis is uncomplicated.
Aug. 6, 1987	DX 6	Dr. Casis	Mild fibro-emphysematous changes and calcified granuloma. No active lung disease.
Mar. 10, 1991	DX 6	Dr. Dugan	No active lung disease.
Oct. 21, 1995	DX 6	Dr. Doyle	Bilateral chronic inflammatory changes. 1.8 cm nodular density left lower lung base.
Nov. 6, 1995	DX 6	Dr. Dugan	Soft tissue nodular density, left lung base and ill-defined nodule right mid lung. CT scan recommended.
Nov. 20, 1995	DX 8	Dr. Mullinger	Pneumonic infiltrate left lower lung; nodular density right mid lung. Malignancy can not be excluded.
Jan. 17, 1996	DX 9	Dr. Bridges	Focal infiltrate in area of previously noted lesions; ill-defined nodular densities bilaterally; tumor can not be excluded.
Feb. 19, 1996	DX 9	Dr. McElany	Right upper lung scarring and soft tissue opacity left lung.
May 24, 1996	DX 9	Dr. Bridges	Pneumonitis present. Increased infiltrate right upper lung.

Although the radiographic record through 1991 did not reveal the presence of any large pulmonary opacities, Dr. Doyle's identification of a 1.8 cm nodule in the October 21, 1995 chest x-ray and the subsequent interpretations of nodular densities establishes the presence of a large pulmonary opacity in Mr. C.'s lungs during the last year of his life. Consequently, the preliminary requirement of a large pulmonary opacity for the irrebuttable presumption 20 C.F.R. § 718.304 (a) has been established.

Other Medical Evidence

Since Mrs. C. has proven the existence of a large opacity in her husband's lungs, I move to the second adjudicative step and consider other relevant medical evidence prior to making a determination of whether the irrebuttable presumption of death due to pneumoconiosis has been invoked. At this stage, I consider all other medical evidence to determine whether that evidence a) conflicts with or confirms a finding of large pulmonary opacity and b) demonstrates that the

[14]There are two general categories of small opacities defined by their shape: rounded and irregular. Within those categories the opacities are further defined by size. The round opacities are: type p (less than 1.5 millimeter (mm) in diameter), type q (1.5 to 3.0 mm), and type r (3.0 to 10.0 mm). The irregular opacities are: type s (less than 1.5 mm), type t (1.5 to 3.0 mm) and type u (3.0 to 10.0 mm). JOHN CRAFTON & ANDREW DOUGLAS, RESPIRATORY DISEASES 581 (3d ed. 1981).

[15]The following designations apply: A – A reader; B – B reader, and BCR – Board Certified Radiologist. These designations indicate qualifications a person may posses to interpret x-ray film. An "A reader" has either submitted six of his chest x-ray interpretations to the Appalachian Laboratory for Occupational Safety and Health ("ALOSH") or taken an approved ALOSH x-ray classification course. A "B Reader" has demonstrated proficiency in assessing and classifying chest x-ray evidence for pneumoconiosis by successful completion of an examination. A "Board Certified Radiologist" has been certified, after four years of study and examination, as proficient in interpreting x-ray films of all kinds including images of the lungs. *See also* 20 C.F.R. § 718.202(a)(1)(ii).

opacity is associated with pneumoconiosis. In this case, the "other" medical evidence includes a CT scan, a pulmonary biopsy, and medical opinion based on evaluation and treatment.

CT Scan

After Dr. Dugan identified the large pulmonary opacity in November 1995, several diagnostic CT scans were accomplished, including one pulmonary evaluation. In a November 8, 1995 lung CT scan, Dr. Doyle observed a 3.8 cm mass and nodular densities bilaterally in the lung fields. A 1.7 cm mass was in the right upper lung, a 1.8 cm nodule was present in the left middle lung, and a 2.0 cm nodular density was present in the left lower lung. In Dr. Doyle's opinion, the nodules were "suspicious for metastatic disease."[16]

Biopsy

On November 22, 1995, Dr. Sudholt conducted a fibrotic bronchoscopy (DX 8). When Dr. Icaza evaluated the lung tissue sample, he reported chronic inflammation with focal areas of necrosis. Dr. Icaza could not rule out ulcerated carcinoma and deferred to further microscopic assessment. Under the microscope, Dr. Liao identified malignant, non-small cell carcinoma.

Medical Opinion[17]

Dr. Barbara Sudholt
(DX 8 and DX 9)

On November 15, 1995, Dr. Sudholt saw Mr. C. based on a referral due to an abnormal chest x-ray. Mr. C. complained about long term and worsening shortness of breath. Mr. C. worked as a coal miner and in a foundry for 15 years. He also worked 12 years in a lumber yard, where he cut asbestos board. Mr. C. smoked a pack of cigarettes a day for 40 years; he quit smoking two weeks before the visit. A November 6, 1995 chest x-ray showed a density in the mid-right lung and a CT scan revealed emphysematous changes, a suspicious mass in the left lung and a lesion on the liver. When listening to Mr. C.'s lungs, Dr. Sudholt heard scattered rhonchi and crackles. As an initial assessment, Dr. Sudholt indicated Mr. C. was a long term cigarette smoker who might have bronchogenic carcinoma with metastatic disease. The physician recommended further extensive evaluation.

On November 20, 1995, Dr. Sudholt hospitalized Mr. C. for two days to evaluate his pulmonary condition due to an abnormal chest x-ray, shortness of breath and left side pain with movement. Upon physical examination, Dr. Sudholt heard bibasilar rhonchi with a few crackles.

[16]Dr. Doyle also observed a lesion on the liver. Liver CT scans on November 21, 1995 and February 16, 1996 disclosed the presence of possible metastatic lesion, measuring 3 cm on the top of the liver (DX 8 and DX 9). A February 26, 1996 spinal CT scan revealed vertebra deformities possibly associated with metastatic disease (DX 5).

[17]Though Dr. O.W. Pflasterer presented several opinions about Mr. C.'s death which I summarize later, I note the record does not contain any treatment notes by Dr. Pflasterer.

The pulmonary function test indicated a mild pulmonary obstruction.[18] The arterial blood gas study did not show a totally disabling respiratory impairment.[19] During the hospitalization, thoracic and liver CT scans revealed lesions and a lung biopsy was positive for malignant lung cancer. Upon discharge, Dr. Sudholt diagnosed lung carcinoma with liver metastases. She also noted the presence of COPD (chronic obstructive pulmonary disease).

During a follow-up visit on November 29, 1996, Dr. Sudholt again heard crackles with rhonchi on physical examination. Dr. Sudholt discussed her lung cancer diagnosis and indicated the difficulty with effective treatment. The prognosis was "quite poor." Dr. Sudholt referred Mr. C. to an oncologist.

Dr. W. Popovic
(DX 9)

In December 1995, upon referral for metastatic lung cancer, Dr. Popovic initiated chemotherapy. Upon initial physical examination, the lungs were clear. Dr. Popovic reported a cigarette smoking history of 70 to 80 pack years. Dr. Popovic diagnosed stage IV lung cancer.

In the spring of 1996, when Mr. C. presented with pain in his right quadrant, a CT scan located a defect on his spine. A course of radiation therapy provided some relief and by June 1996, Mr. C. seemed to be doing better. However, on September 24, 1996, Mr. C. returned with renewed abdominal pain. His liver appeared enlarged and he was failing rapidly. Mr. C. was referred to a hospice.

Dr. P. Kurichety
(DX 9)

Between December 19, 1995 and January 9, 1996, Dr. Kurichety provided irradiation therapy for Mr. C.'s spinal lesion and in an effort to ease his breathing difficulties. Mr. C. had been diagnosed with left lung carcinoma with hepatic metastatic disease. In response to abdominal pain in March 1996, Dr. Kurichety provided another round of irradiation therapy to the spinal lesion.

Discussion

The November 8, 1995 CT scan finding of a 3.8 cm mass clearly confirmed the radiographic finding of a large pulmonary opacity. However, both the biopsy evidence and medical opinion demonstrate that the large pulmonary opacity involved lung cancer. Notably, no objective medical evidence indicates the large pulmonary opacity was pneumoconiosis. Accordingly, I find Mr. C. did not have complicated pneumoconiosis and Mrs. C. is unable to invoke the irrebuttable presumption of death due to pneumoconiosis under 20 C.F.R. § 718.304.

[18]The November 21, 1995 pulmonary function test produced the following results: pre-bronchodilator, FEV_1 – 1.55 and FVC 3.10; post-bronchodilator, FEV_1 – 1.59 and FVC 3.23.

[19]In the November 21, 1995 arterial blood gas study, the pCO_2 was 33 and the pO_2 was 75.7. Under 20 C.F.R. § 718, Appendix C, the qualifying pO_2 is 67.

Death Caused by Pneumoconiosis

As set out in Mr. C.'s death certificate, DX 4, all the physicians who considered Mr. C.'s demise concluded that metastatic lung cancer was the direct cause of his death. Correspondingly, no physician stated that Mr. C.'s death was directly caused by the coal workers' pneumoconiosis. Accordingly, I find Mr. C.'s death was not caused by pneumoconiosis.

Death Caused by Complications of Pneumoconiosis

Although Mr. C.'s death was not directly caused by lung cancer, Mrs. C. may still be entitled to survivor benefits if complications from pneumoconiosis contributed to his death. In the presentation of her claim, this potential manner of recovery is predicated on the assertion that Mr. C.'s lung cancer was a complication of his exposure to coal mine dust which contained silica.

In determining whether a relationship existed between Mr. C.'s lung cancer and his coal workers' pneumoconiosis and exposure to coal mine dust, I will consider the following statements from friends and co-workers, a coal sample report for the Illinois Department of Natural Resources, a brief summary of medical studies, the 2001 findings of the U.S. Department of Health and Human Services, and medical opinion.

Mr. Dorcas Gruber
(DX 39)

In an August 8, 2000 statement, Mr. Gruber stated that he worked with Mr. C. for 12 years in a lumber yard. During the period, since contractors preferred to do their own cutting, Mr. C. seldom cut asbestos board.

Mrs. Mary Belton
(DX 39)

According to Mrs. Belton, her husband worked with Mr. C. for 15 years in a magnesium foundry. Her husband reported that the work was hot and dusty. Both men received several burns from the hot metal. According to Mrs. Belton, the work was "always hot and dusty with silica sand covering everything." Silica flour was used to coat the molds to facilitate removal of the castings upon cooling. Mrs. Belton was aware that Mr. C. worked long shifts because "he went in early to fire the coal furnaces to get them hot enough to melt the metal." During this process, he was exposed to furnace fumes. Mr. C. experienced additional silica exposure during routine maintenance and repair of the furnaces "because they used silica-rich refractory materials in the furnaces and ladles."

Mr. C. Arthur Rice
(DX 39)

Mr. Rice, an administrator for the Illinois Department of Natural Resources, reported that coal samples from a mine identified by Mrs. C.'s lay representative that operated in the late 1940s contained silica dioxide in the form of quartz. Typically, during that era, the coal mines did not take any dust mitigation measures.

Medical Articles and Studies[20]
(DX 13, DX 15, DX 28, DX 34, DX 49, and CX 1)

As Judge Hillyard observed, in support of her claim, Mrs. C. submitted "voluminous medical studies," totaling over 400 pages and covering reports from 1936 to 2001. This mass of scientific data leads to some generalizations. First, cigarette smoking and exposure to coal mine dust can cause respiratory dysfunction. Second, in early studies the role of silica in the development of emphysema was unclear. Third, exposure to silica continues to be a health risk in coal miners. Fourth, some studies were in conflict as the relationship between coal mine employment and the development of lung cancer since some increase may be due to differences in smoking habits. Fifth, a significant relationship exists between the length of underground coal mining and the prevalence and severity of silicosis. Sixth, silicosis is more strongly associated with higher levels of coal workers' pneumoconiosis. Seventh, a significant number of coal miners receive a high enough concentration of free silica to produce silicosis. Eighth, while exposure to excessive amounts of respirable coal mine dust increases the risk for the development of progressive massive fibrosis, chronic bronchitis, and emphysema, at least one study did not find a similar association with between coal mine dust exposure and lung cancer. Ninth, coke oven emissions are known to be human carcinogens.

U.S. Department of Health and Human Services Publications
(DX 34 and DX 39)

In a September 1995 publication on occupational exposure to respirable coal mine dust, based on various medical studies, the U.S. Department of Health and Human Services ("HHS") described the three types of silicosis that may develop from inhaled respirable crystalline silica. Chronic silicosis "commonly involves 15 or more years of exposure to silica" and accelerated silicosis occurred with an exposure of 5 to 10 years. The third type, acute silicosis, usually arose within 6 months to 2 years of "intensive exposure to fine particles of nearly pure silica – such as those present during sandblasting or drilling."

In January 2001, HHS revised a report on known human carcinogens. The report noted the increased presence of respirable crystalline silica, primarily quartz dust, in several industries, including coal mining and foundries. In coal mining, silica was found in coal mine dust produced during operations to extract coal. In foundries, silica sand and flour was used in molds. Based on several studies, and even accounting for other factors such as cigarette smoking and

[20]Standing alone, the medical studies are not probative on the issue of whether Mr. C. died due to pneumoconiosis. However, while clearly not as comprehensive as the parties may prefer, a summarization helps puts into perspective the opinions of the physicians who reviewed the studies in conjunction with Mrs. C.'s survivor claim.

asbestos exposure, HHS concluded elevated risk for cancer was associated with the occupationally related silica exposure. Consequently, the exposure levels to respirable crystalline silica is regulated by EPA, FDA, and OSHA.

Dr. John E. Myers, Jr.
(DX 34)

On June 13, 1998, Dr. Myers reviewed Mr. C.'s medical record and deposition. Mr. C. worked as a teenager in a coal mine about over 4 years picking slate at the tipple, loading coal, and working other labor jobs. He also worked 15 years in a foundry in the furnace area, melting metal into molds. Finally, Mr. C. was employed an additional 20 years in a lumber yard. For most of his life, Mr. C. smoked a pack of cigarettes a day. Although the lumber yard work did not involve any significant pulmonary risks, Mr. C. was exposed to silica dioxide in his foundry work. The radiographic evidence was consistent with pneumoconiosis or silicosis and the pulmonary tests met the total disability thresholds.

Based on his review, Dr. Myers identified three pulmonary health risks: cigarette smoking, exposure to silica dioxide at the foundry, and coal mine dust exposure. Standing alone, other than work drilling coal or working a continuous miner, four and a half years of coal mine employment is "rarely a cause of significant disability or radiographic evidence of silicosis." However, when "one adds cigarette usage and foundry work to the four plus years in coal mines," a "sufficient history of exposure" is established to support a diagnosis of silicosis/chronic obstructive pulmonary disease." In Dr. Myers' opinion, Mr. C.'s pneumoconiosis and pulmonary disability were related to all three etiologies. At the same time, Mr. C.'s cigarette smoking was more significant than his foundry work which in turn was more significant than his coal mine employment.

Dr. Sarah B. Long
(DX 16)

On September 23, 1997, Dr. Long reviewed the medical studies (DX 13 and DX 15) submitted by Mrs. C. in support of her claim. In her opinion, the articles did not "clearly link coal workers' pneumoconiosis with carcinoma of the lung." She noted that some of the studies even showed a greater incidence of lung cancer among cigarettes smokers in comparison to non-smokers. Additionally, several of the reports were old and did not reflect current literature. Current studies have not shown a significant link between coal workers' pneumoconiosis and lung cancer. Dr. Long opined that Mr. C.'s long term cigarette smoking history was the most likely cause of his lung cancer.

Dr. Leon Cander
(DX 23 and DX 48)

In March 1998, Dr. Cander, board certified in internal medicine, reviewed Mr. C.'s medical record. Dr. Cander noted that Mr. C. worked less than five years as a coal miner and had a cigarette smoking history of 75 pack years. During the course of his life, Mr. C. also worked in a magnesium factory with exposure to silica and had been exposed to asbestos in the

workplace. During the last year of his life, none of the radiographic studies were reported positive for pneumoconiosis and the November 1995 pulmonary function and respiratory studies did not exceed the regulatory thresholds for total disability. In regards to medical studies, Dr. Cander stated that several reports showed cigarette smoking was "the most powerful lung cancer carcinogen." While coal mine dust can cause COPD, no definitive medical study has established a causation link between COPD – lung cancer, and coal mine dust – lung cancer. Additionally, no cause and effect relationship had been established between silica exposure and lung cancer. Since no autopsy was performed, no definitive evidence of pulmonary scarring associated with silicosis or silica exposure exists in Mr. C.'s case. In Dr. Cander's opinion, Mr. C. had metastatic lung cancer caused by the "most powerful combination" of two powerful carcinogens: 75 pack years of cigarette smoking and asbestos exposure.

In June 2003, Dr. Cander again addressed the applicability of medical studies to Mr. C.'s case. While one study had shown a connection between dust exposure in coke oven workers and lung cancer, Mr. C. did not operate a coke oven and wasn't exposed to those toxic fumes. At best, due to known deficiencies associated with "confounding" exposure to other environmental carcinogens, such as cigarette smoke, medical studies only show a "suspected" connection between silica/silicosis and lung cancer. No definitive connection has been established. Studies since 1996 still failed to show a carcinogenic effect of silicosis.

Dr. David F. Goldsmith
(DX 34)

On February 25, 2000, Dr. Goldsmith, who holds a PhD in epidemiology, observed that in both his five years as an underground coal miner and fifteen years as a foundryman, Mr. C. suffered extensive exposure to silica, which is known to cause silicosis and pneumoconiosis which are subsets of COPD. The cumulative twenty years of silica exposure explains Mr. C.'s pulmonary condition. Summarizing an extensive number of studies, including many of his works, Dr. Goldsmith opined that a "large body of literature" demonstrates "a link between silica exposure, silicosis, and lung cancer." Additionally, silica exposure and cigarette smoke act synergistically to enhance the risk of lung cancer. Based on his review of the medical record, Dr. Goldsmith concluded Mr. C. had silicosis, coal workers' pneumoconiosis or COPD from 1973 until he was diagnosed with non-small cell lung cancer. Consequently, "Mr. [C]'s fibrotic lung disease from exposure to silica dust was more likely than not the cause of his lung cancer." Because Mr. C.'s chronic lung disease from 1973 preceded the development of lung cancer, that initial pulmonary condition was a significant cause of his death from lung cancer.

Dr. Jeffrey D. Britton
(DX 49)

On May 21, 2003, Dr. Britton, board certified in preventive medicine and occupational medicine, reviewed Mr. C.'s medical record, interpreted a September 1985 chest x-ray, reviewed numerous medical studies, and considered reports from several physicians and experts, including Dr. Cander, Dr. Long, and Dr. Goldsmith. Mr. C. worked 4 and 3/4 years in a coal mine with "heavy exposure to coal and silica as per the letter of the State of Illinois." During his 15 years in a foundry, Mr. C. also received significant exposure to silica and coal combustion gases and

possibly asbestos. Mr. C. had an "extensive" cigarette smoking history of over 40 years, equating to about 40 pack years. After noting that the chest x-ray established the presence of uncomplicated pneumoconiosis or silicosis, Dr. Britton specifically noted that contrary to Dr. Cander's presentation, the most recent peer review publications have established a causal association between silica exposure and lung cancer. Many government agencies, including NOISH, now recognize respirable silica as a known human carcinogen. Based on Mr. C.'s exposure to known pulmonary occupational hazards, Dr. Britton attributed the development of lung cancer to the "synergistic activity of cigarette smoking, the effect of coal dust, [and] the effects of coal dust, silica, coal combustion and asbestos."

Discussion

In addressing this particular causation issue in Mrs. C.'s survivor claim, I must determine whether a) Mr. C. had coal mine-related silicosis, b) silicosis can lead to the development of lung cancer, and c) Mr. C.'s lung cancer was related to silicosis arising out of his coal mine employment.

Coal Mine-Related Silicosis

Regarding the presence of silicosis in Mr. C.'s lungs, while the radiographic record showed the presence of opacities consistent with pneumoconiosis, no physician definitively diagnosed silicosis. At best, Dr. Britton rendered an equivocal finding by stating the opacities represented pneumoconiosis or silicosis. Further, notably absent in Mr. C.'s treatment records, pulmonary evaluations, and lung biopsy is any diagnosis of silicosis.

Even if Mr. C. had silicosis, the record does not clearly establish its source. During the course of his life, Mr. C. was exposed two principal sources of silica. As recognized by most physicians, and supported by the statement of Mrs. Belton, Mr. C.'s most significant and extensive exposure to silica occurred during his 15 years as a foundryman in a magnesium casting plant. At the same time, coal samples from the state of Illinois demonstrate that Mr. C. was also exposed to silica during his nearly five years as a coal miner. Although some doctors opined that Mr. C.'s disabling pulmonary disease was due to a combination of two occupations, Dr. Myers also acknowledged that four and a half years of coal mine employment "rarely" causes a pulmonary impairment unless the miner drilled coal or operated a continuous miner. Similarly, though one medical study indicated that acute silicosis may arise in as little 6 months to two years in extremely dusty conditions, that finding was specifically limited to sandblasting and drilling. The record does not indicate that Mr. C. drilled coal or operated a miner. Instead, according to Dr. Myers, Mr. C. worked as a slate picker, coal loader, and laborer.

Significantly, this inconclusive evidentiary record concerning whether Mr. C. had coal mine-related silicosis is overcome by a stipulation entered into by the parties at the October 2005 hearing. As previously noted, the parties have stipulated that Mr. C. had coal workers' pneumoconiosis. Under the regulation, 20 C.F.R. § 718.201(a)(1), the definition of pneumoconiosis includes "silicosis." Based on this definition and the addition of "coal workers'" in the stipulation, the parties have essentially agreed that Mr. C. had silicosis that arose at least in part out of his coal mine employment.

Relationship Between Silicosis and Lung Cancer

As demonstrated by the dozens of medical studies and articles contained in this claim, the relationship between coal mining pulmonary hazards and the development of lung cancer has been extensively evaluated.

According to Dr. Long, the initial medical studies submitted by Mrs. C. did not demonstrate a link between coal mining and lung cancer. Similarly, Dr. Cander opined the medical studies do not show a definitive connection between exposure to silica/coal mine dust and lung cancer. Disagreeing, Dr. Goldsmith found the medical studies established a causation association between silicosis and lung cancer. Agreeing with Dr. Goldsmith, Dr. Britton emphasized that recent peer review studies have determined that occupational silica is a cancer causing agent.

In assessing the respective probative value of this expert standoff, I give diminished probative value to opinions of Dr. Long and Dr. Cander due to the dated nature of the studies upon which they relied. In concluding no conclusive causation connection existed, Dr. Long and Dr. Cander reasonably highlighted the conflicting and uncertain findings arising out of the earlier medical studies on the link between silica exposure and lung cancer. However, as noted by Dr. Britton, more recent medical studies have definitively established that connection. Additionally, the opinions of Dr. Goldsmith and Dr. Britton are further support by HHS' recognition in 2001 that respirable silica found in many industries, including coal mining, is a known human carcinogen which increases the risk of lung cancer.

Mr. C.'s Silicosis and Lung Cancer

Since Mr. C. had silicosis arising in part out of his coal mine employment, and the more probative opinions indicate silica exposure increases the risk for lung cancer, I finally turn to the issue of whether Mr. C.'s terminal lung cancer was a complication of his coal workers' pneumoconiosis.

Based on her assessment that medical studies did not establish a sufficient connection, Dr. Long concluded Mr. C.'s lung cancer developed as a result of his extensive cigarette smoking. Relying in part on the absence of medical study validation, and noting the absence of any objective medical evidence showing Mr. C. had silicosis, Dr. Cander concluded Mr. C.'s lung cancer was due to his extensive cigarette smoking history and exposure to asbestos. Dr. Goldsmith opined the two significant carcinogen risks of cigarette smoke and silica coal mine dust worked together to increase Mr. C.'s risk of developing lung cancer. Dr. Britton also believed the combination of cigarette smoking and exposure to silica, including silica coal mine dust, acted synergistically to cause Mr. C.'s lung cancer.

In sorting through this difference of opinion, I give diminished probative weight to Dr. Long's conclusion which is based on a premise inconsistent with my determination that the more probative medical opinion establishes a connection between coal mine silica exposure and lung cancer. Dr. Cander's opinion loses probative value for the same reason and due to his conclusion that Mr. C. did not have silicosis, which is also contrary to my determination. Next, although

holding a PhD in epidemiology, Dr. Goldsmith is not a physician. As a result, his conclusion about the specific nature and cause of Mr. C.'s death has diminished probative value.

Finally, turning to the pivotal medical opinion on this issue, I find that Dr. Britton's attribution of Mr. C.'s lung cancer in part to his coal-mine related silicosis has insufficient probative value to support a finding that Mr. C.'s lung cancer was in part a complication of his silicosis. Although Dr. Britton understandably relies on medical studies showing an increased risk of lung cancer due to exposure to silica exposure, the physician is unable to identify any objective medical evidence specific to Mr. C.'s lung cancer that demonstrates that Mr. C. fell victim to that increased risk.[21] As noted by Dr. Myers decades ago, Mr. C. faced three pulmonary hazards in descending order of significance: a 40 pack year history of cigarette smoking, 15 years of significant silica dust exposure in a magnesium foundry, and 4 and 3/4 years of significant exposure to coal mine dust containing respirable silica. In concluding that all three hazards acted synergistically in Mr. C.'s case, Dr. Britton essentially relies on medical studies which identify all three agents as carcinogens without commenting on whether the two more pronounced risk exposures associated with cigarette smoking and foundry work, either standing alone or even together, might just as likely explain Mr. C.'s lung cancer. Such plausible alternative causation conclusions highlight the necessity for objective medical evidence specific to Mr. C.'s lung cancer to support a finding that his lung cancer was caused in part by 4 and 3/4 years of coal mining.

In summary, through a stipulation of fact, Mrs. C. has established that Mr. C. had silicosis related to coal mine employment. Additionally, the preponderance of the more probative medical opinion establishes that silica exposure increases the risk of lung cancer. However, for various reasons, the medical evidence and opinion in the record is insufficient to establish that Mr. C. developed lung cancer in part due to his silica exposure while working as a coal miner. Consequently, Mrs. C. is unable to prove that her husband died as a result of a complication of coal workers' pneumoconiosis/silicosis.

Pneumoconiosis Was a Substantially Contributing Cause of, or Hastened, Death

Even though neither pneumoconiosis nor its complications caused Mr. C.'s death, his spouse may still be entitled to survivor benefits if pneumoconiosis was a substantially contributing cause of her husband's death. Prior publication of the new regulations, the U.S. Court of Appeals for the Fourth Circuit interpreted "substantially contributing cause" to include a hastening of a miner's death in any way. *Shuff v. Cedar Coal Co.*, 967 F.2d 977, 980 (4th Cir. 1992); *Richardson v. Director, OWCP*, 94 F.3d 164 (4th Cir. 1996). The U.S. Court of Appeals for the Seventh Circuit, which has jurisdiction over this claim, applies the same standard. *See Peabody Coal Co. v. Director, OWCP,* 972 F.2d 178 (7th Cir. 1992). Adopting that standard, the new regulation, 20 C.F.R. § 718.205(c)(5), states "pneumoconiosis is 'a substantially contributing cause' of a miner's death if it hastens the miner's death." Under this legal standard, if pneumoconiosis cut short Mr. C.'s life in any manner, Mrs. C. may prevail with her modification request and survivor claim.

[21]While obviously hardly representative of the entire lungs, the detailed analysis and assessment of the November 1995 lung biopsy, which led to a diagnosis of non-small cell carcinoma, did not disclose the presence of cells associated with either pneumoconiosis or silicosis (DX 9).

Dr. O.W. Pflasterer
(DX 6, DX 13, DX 22, and DX 34)

In December 1996, Dr. Pflasterer indicated that Mr. C. had been an underground coal miner and developed coal workers' pneumoconiosis. In Dr. Pflasterer's opinion, Mr. C. died "due to carcinoma of the lung with hepatic and osseous metastasis."

On May 30, 1997, Dr. Pflasterer stated that he treated Mr. C. from January 1972 to October 4, 1996. Dr. Pflasterer found consistent evidence of emphysema and fibrosis. Mr. C. was awarded black lung disability benefits. In November 1995, Mr. C. was diagnosed with carcinoma of the lungs "with local spread and hepatic metastasis" and "expired as a result of that condition" on October 4, 1996. In Dr. Pflasterer's opinion, Mr. C.'s pneumoconiosis "was a likely contributing factor to his demise."

On October 7, 1997, Dr. Pflasterer observed that from the beginning of his treatment, Mr. C. had progressively developing pulmonary emphysema and fibrosis. Dr. Pflasterer again noted Mr. C. had been awarded black lung disability benefits. In Dr. Pflasterer's opinion, due to the "additional adverse effect on his pulmonary function status, the chronic lung condition contributed to, and hurried Mr. [C.'s] demise due to non-small cell lung carcinoma."

On October 20, 1998, Dr. Pflasterer stated that Mr. C.'s underlying disease of pneumoconiosis, "with decreased baseline pulmonary function reserve did in fact hasten his demise."

Coroner Neil Birchler
(DX 4 and DX 34)

On October 17, 1996, Corner Birchler signed Mr. C.'s death certificate. Noting the attending physician as Dr. O.W. Pflasterer, Coroner Birchler indicated Mr. C. died due to carcinoma of the lung with metastasis. Another significant condition contributing to death was "essential vascular hypertension."

On February 16, 2000, advised by Dr. Pflasterer that Mr. C. had coal workers' pneumoconiosis and was receiving black lung disability benefits, Coroner Birchler revised the death certificate. As a significant condition contributing to death, Coroner Birchler added "coal miner's pneumoconiosis."

Dr. Leon Cander
(DX 23 and DX 48)

In March 1998, based on his review of the medical record, Dr. Cander observed that although Mr. C. was receiving black lung disability benefits based on COPD attributable to coal mine employment, the November 1995 pulmonary studies showed only a mild pulmonary obstructive impairment, which would not have caused or contributed to Mr. C.'s death.

On June 23, 2003, Dr. Cander again reviewed Mr. C.'s case and specifically considered Dr. Britton's assessment. Dr. Cander again stressed that the November 1995 pulmonary tests, in particular the arterial blood gas study, indicated the absence of hypoxemia which is usually major contributing factor in death associated with coal workers' pneumoconiosis. Absent that contributing factor, Mr. C.'s death was due solely to hepatic, pulmonary, and bone metastases from primary lung cancer.

Dr. Jeffrey D. Britton
(DX 49)

Based on his extensive review of the medical record, Dr. Britton indicated Mr. C. died due to lung cancer produced by the synergistic combination of several pulmonary risk including cigarette smoking and exposure to silica and coal mine dust. These well known pulmonary hazards caused a pulmonary impairment, and contributed to and hastened Mr. C.'s death.

Discussion

Dr. Pflasterer, Dr. Britton, and Coroner Birchler concluded Mr. C.'s coal workers' pneumoconiosis was a contributing cause of death. Dr. Cander disagreed.

In resolving this dispute, I first give little probative weight Coroner Birchler's revised death certificate because he provided no independent rationale for his determination. When Coroner Birchler added coal workers' pneumoconiosis as a contributing factor of death, he did so based solely on Dr. Pflasterer's statement that Mr. C. had black lung disease and was receiving black lung disability benefits.

Next, for two reasons, I also give Dr. Cander's opinion diminished probative weight. First, Dr. Cander relied on pulmonary tests conducted about a year before Mr. C.'s death which indicated only a mild obstruction and the absence of hypoxemia. However, the reliability of those tests can be questioned since they were conducted during Mr. C.'s hospitalization in November 1995 and do not conform to the regulatory requirements. Second, Dr. Cander's reliance on the absence of a significant impairment is inconsistent with the prior determination in Mr. C.'s claim that he was totally disabled due to coal workers' pneumoconiosis.

Dr. Britton's finding on this issue also suffers a loss of probative value. As previously discussed due to the absence of any objective medical evidence specifically tying Mr. C.'s lung cancer to his coal mine dust exposure, Dr. Britton has to rely on the insufficient basis of a presumed synergistic effect of all three pulmonary hazards in the hastening of Mr. C.'s death.

In contrast, Dr. Pflasterer provided a documented and reasoned determination that Mr. C.'s coal workers' pneumoconiosis hastened his death through a reduction in his pulmonary reserve. In terms of documentation, although his treatment notes are not in the record, Dr. Pflasterer provided numerous radiographic studies in support of Mrs. C.'s survivor claim (DX 6). He also ordered the November 1995 chest x-ray that lead to the diagnostic lung cancer referral to Dr. Sudholt. Several of the subsequent radiographic studies, CT scans, and medical reports in the record, including the oncology and radiation treatment summaries, list Dr. Pflasterer as the

treating/referring physician or indicate that he was sent a copy of the report. Based on that documentation, and after stating that Mr. C. died to lung cancer with hepatic and osseous metastasis, Mr. C.'s treating physician notes that he struggled with pulmonary insufficiency since the early 70s. Dr. Pflasterer emphasizes that eventually Mr. C.'s pulmonary defect lead to an award of black lung disability benefits. Dr. Pflasterer then reasonably explained that due to the established pulmonary disability caused by the underlying disease of pneumoconiosis, Mr. C. had a decreased pulmonary reserve that directly hastened his death.

In summary, due to various reasons, the assessments of Dr. Cander, Coroner Birchler, and Dr. Britton have diminished probative value. The remaining opinion by Dr. Pflasterer remains sufficiently probative to establish that coal workers' pneumoconiosis hastened Mr. C.'s death due to metastatic lung cancer.

Summary

Through probative medical opinion, Mrs. C. has proven that coal workers' pneumoconiosis hastened Mr. C.'s death. Correspondingly, she has demonstrated that a modification of the denial of her second modification request is warranted. That modification effectively revives her survivor claim. As a result, I must again consider the entire record to determine whether Mrs. C. is entitled to survivor benefits.

Issue #2 – Entitlement to Survivor Benefits

As previously discussed, a survivor claim has four elements of entitlement. First, the claimant must be an eligible survivor. Second, the deceased coal miner had pneumoconiosis. Third, the deceased coal miner's pneumoconiosis must have arisen, at least in part, out of coal mine employment. Fourth, the coal miner's death was due to coal workers' pneumoconiosis.

In Mrs. C.'s case, the first three elements of entitlement were established through stipulations. Specifically, Mrs. C. is an eligible survivor under Act; Mr. C. had pneumoconiosis; and, his pneumoconiosis arose out of his coal mine employment. The fourth element of entitlement has been established through my adjudication of Mrs. C.'s third modification request and determination that Mr. C.'s death was hastened by coal workers' pneumoconiosis. Accordingly, having proven that Mr. C.'s death was due to coal workers' pneumoconiosis, Mrs. C. has met her burden of proof and her survivor claim must be approved.

DATE OF ENTITLEMENT

Since the modification in this case was based on a mistake of fact and involves a survivor claim, 20 C.F.R. §§ 725.503(c) and (d)(1) establish the date of entitlement as the beginning of the month the coal miner died due to pneumoconiosis. Since Mr. C. passed away on October 4, 1996, Mrs. C.'s survivor benefits are payable beginning October 1, 1996.

ORDER

The claim of MRS. E.M.C. for survivor benefits under the Act is **GRANTED**. Benefits shall commence October 1, 1996.

SO ORDERED:

A

RICHARD T. STANSELL-GAMM
Administrative Law Judge

Date Signed: November 17, 2006
Washington, DC

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. See 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. See 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. See 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).